CLASSIFICATION RESTRICTED
SECURITY INFORMATION
CENTRAL INTELLIGENCE AGENCY

INFORMATION FROM
FOREIGN DOCUMENTS OR RADIO BROADCASTS

REPORT

CD NO.

STAT

COUNTRY	Yugoslavia	DATE OF INFORMATION	1953
SUBJECT	Scientific - Medicine, effects of cold		
HOW PUBLISHED	Irregular periodical	DATE DIST. 14	Sep 1953
WHERE PUBLISHED	Belgrade	NO. OF PAGES	2
DATE PUBLISHED	1953		
LANGUAGE	French	SUPPLEMENT TO REPORT NO.	

THIS IS UNEVALUATED INFORMATION

SOURCE Acta Medica Iugoslavica, Vol VII, No 1-2, pp 40-62.

DEATH MECHANISM OF WARM-BLOODED ANIMALS WHO HAVE DIED AS A RESULT OF EXPOSURE TO LOW TEMPERATURES

V. Popovic
Inst of Physiol, Faculty of Sci
Belgrade Univ

[The following abstract of Popovic's article is taken from Acta Medica Iugoslavica, Vol VII. No 1-2, 1953.]

In this article the author elaborates on the limits within which life can be preserved in experimentally induced hypothermia. He discusses the levels of refrigeration from which a warm-blooded animal may recover, and also the mechanism of death caused by extreme cold.

1. The bodies of rats and other mammals may be reduced in temperature to about 14°-15° (rectal temp), with subsequent restoration to their normal condition, providing that the state of deep hypothermia (lowering of the temperature to 14°) has not exceeded 10-12 hrs.

2. By applying artificial respiration, rats, guinea pigs, rabbits, and probably other mammals may be safely cooled to temperatures of 10°, 9°, and even low as 6°. By means of artificial reheating, the temperature of the experimentally cooled animals may be brought back to a normal level. In the majority of cases, the animals will easily revert to their normal state. Consequently, cooling of the body below 15° is not lethal, contrary to the assumption of certain authors. This temperature is not "the lethal temperature of tissues" as Adolph maintains.

3. The curve of the dissociation of oxyhemoglobin at low temperatures in vitro (according to Barcroft) does not seem to apply in vivo. The oxyhemoglobin is not stable in deep hypothermia. It continues to provide the tissues with oxygen, even though the temperature of the blood drops below 15°.

Thus, small doses of CO, which as a rule reduce the consumption of O_2 only slightly in normal animals, lower to a considerable degree the consumption of this gas in rats whose temperature has been brought down to 15°. Doubtlessly the carbon monoxide replaces the oxygen content of oxyhemoglobin.

On the other hand, the increase in the pressure of O_2 in the plasma caused by placing the refrigerated animal into pure oxygen does not increase the animal's consumption of oxygen beyond that which would have occurred in a normal atmosphere or under a slightly reduced pressure. The limiting pressure of oxygen, i.e., the stage at which progressive depression begins to affect the respiratory tract, is equal to about 80-100 mm Hg both in a normal rat and in a rat cooled to 15°. The effect of various factors can increase to a considerable extent the oxygen consumption of a rat whose temperature has been lowered. This consumption can be also stimulated by such factors as feeding the animal meat, adaptation to the cold, etc.

All these findings disprove as hardly plausible the hypothesis that death through lowering of temperature is due to an asphyxiation resulting from a stoppage of the supply to the tissues of oxygen contained in the oxyhemoglobin. This is a theory defended by numerous physiologists (see Verz, Weltz, Lutz, Schumacher, etc.)

4. The "theory of the heart," which explains death caused by extreme cold as a direct effect of the cold on the heart, by overtaxation of the heart, anoxemia of the heart, etc. (Bigelow), seems hardly justified. An extirpated heart continues to beat even after its temperature has been reduced below 15° (down to 6°). A venesection relieves the strain on the right side of the heart, but does not decrease the body temperature of a warm-blooded animal to its lethal limits. The heart of an animal brought to a temperature below 15° and as low as 8°-10° functions much better (pulsation increases from 10-12 to 40-60 beats per minute) if artificial respiration is applied.

5. Moreover, one may refute the theory that death from extreme cold is caused by paralysis of the nervous system. It is evident that all reflexes disappear at a temperature only a few degrees higher than 15°. However, reduction of the temperature of animal bodies to a temperature below 15° and down to 6°-8°, with the application of artificial respiration, is accompanied by the retention of certain reflexes. This means that the disappearance of reflexes at a body temperature of 15° is not the cause but the result of the general condition of the animal and is induced by the faulty functioning of the respiratory center. I.e., the disappearance of reflexes is due to asphyxiation.

6. One must regard the disorders of the respiratory systems as the principal cause of the death of warm-blooded animals whose body temperature has been lowered to 15°. The respiratory disturbances can be attributed to a reduced excitability of the respiratory centers. This assumption is supported by the following findings: At 15°, respiration ceases before the heart stops. Approximately 3% of CO_2 in the air inhaled by the refrigerated animal produces a favorable effect on the general condition of this animal and assists in its recovery. This phenomenon has been observed by Gelhorn and Janus (1936), who had stated that "the animals then are in the best condition." Consequently, for use in the artificial respiration administered to animals affected by cold, the following mixture of gases is recommended:

37% of O_2 plus 3% of CO_2 [plus 60% of N_2 or of air]

- E N D -